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  * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 10:50:18 ON 06 JUN 2003

=> fil .bec
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

FILES 'MEDLINE, SCISEARCH, LIFESCI, BIOTECHDS, BIOSIS, EMBASE, HCAPLUS, NTIS,
       ESBIOBASE, BIOTECHNO, WPIDS' ENTERED AT 10:50:38 ON 06 JUN 2003
ALL COPYRIGHTS AND RESTRICTIONS APPLY.  SEE HELP USAGETERMS FOR DETAILS.

11 FILES IN THE FILE LIST

=> s dna(w)(pk or activated(w)(protein kinase# or pk))
FILE 'MEDLINE'
        694237 DNA
        134256 PK
        190484 ACTIVATED
       1161323 PROTEIN
        190849 KINASE#
         92078 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
        134256 PK
L1         680 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'SCISEARCH'
        480933 DNA
         16920 PK
        202879 ACTIVATED
        995241 PROTEIN
        213447 KINASE#
        107420 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
         16920 PK
L2         524 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'LIFESCI'
        229692 DNA
          4313 PK
         71872 ACTIVATED
        403015 "PROTEIN"
         62069 KINASE#
         30432 PROTEIN KINASE#
                 ("PROTEIN"(W)KINASE#)
          4313 PK
L3         334 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'BIOTECHDS'
         99133 DNA
           251 PK
         10507 ACTIVATED
        101746 PROTEIN
          6520 KINASE#
          1168 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
           251 PK
L4           6 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'BIOSIS'
        701206 DNA
         14718 PK
        219476 ACTIVATED
```

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       1297795 PROTEIN
        243329 KINASE#
        112764 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
         14718 PK
L5         575 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'EMBASE'
        511309 DNA
        146991 PK
        190545 ACTIVATED
       1113247 "PROTEIN"
        164715 KINASE#
         84253 PROTEIN KINASE#
                 ("PROTEIN"(W)KINASE#)
        146991 PK
L6         474 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'HCAPLUS'
        612489 DNA
         20957 PK
        407484 ACTIVATED
       1516037 PROTEIN
        200733 KINASE#
         93976 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
         20957 PK
L7         538 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'NTIS'
          8508 DNA
           362 PK
          9722 ACTIVATED
         11926 PROTEIN
          1366 KINASE#
           449 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
           362 PK
L8          10 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'ESBIOBASE'
        207714 DNA
          6175 PK
         86664 ACTIVATED
        458809 PROTEIN
         85258 KINASE#
         49964 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
          6175 PK
L9         431 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'BIOTECHNO'
        363454 DNA
          4472 PK
         85314 ACTIVATED
        572356 PROTEIN
         83617 KINASE#
         43669 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
          4472 PK
L10        351 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

FILE 'WPIDS'
         48719 DNA
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          1508 PK
        115820 ACTIVATED
         98077 PROTEIN
          7134 KINASE#
          1939 PROTEIN KINASE#
                 (PROTEIN(W)KINASE#)
          1508 PK
L11         17 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

TOTAL FOR ALL FILES
L12       3940 DNA(W)(PK OR ACTIVATED(W)(PROTEIN KINASE# OR PK))

=> s l12 not 1994-1996/py
FILE 'MEDLINE'
       1244236 1994-1996/PY
L13        581 L1 NOT 1994-1996/PY

FILE 'SCISEARCH'
       2559592 1994-1996/PY
L14        438 L2 NOT 1994-1996/PY

FILE 'LIFESCI'
        325401 1994-1996/PY
L15        275 L3 NOT 1994-1996/PY

FILE 'BIOTECHDS'
         46905 1994-1996/PY
L16          6 L4 NOT 1994-1996/PY

FILE 'BIOSIS'
       1660749 1994-1996/PY
L17        485 L5 NOT 1994-1996/PY

FILE 'EMBASE'
       1117373 1994-1996/PY
L18        398 L6 NOT 1994-1996/PY

FILE 'HCAPLUS'
       2256014 1994-1996/PY
L19        461 L7 NOT 1994-1996/PY

FILE 'NTIS'
        131755 1994-1996/PY
L20          8 L8 NOT 1994-1996/PY

FILE 'ESBIOBASE'
        498223 1994-1996/PY
L21        367 L9 NOT 1994-1996/PY

FILE 'BIOTECHNO'
        304425 1994-1996/PY
L22        291 L10 NOT 1994-1996/PY

FILE 'WPIDS'
       1732428 1994-1996/PY
L23         17 L11 NOT 1994-1996/PY

TOTAL FOR ALL FILES
L24       3327 L12 NOT 1994-1996/PY

=> s l24 not 1997-1999/py
FILE 'MEDLINE'
       1333570 1997-1999/PY
L25        317 L13 NOT 1997-1999/PY
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FILE 'SCISEARCH'
       2862496 1997-1999/PY
L26         240 L14 NOT 1997-1999/PY

FILE 'LIFESCI'
        337226 1997-1999/PY
L27         130 L15 NOT 1997-1999/PY

FILE 'BIOTECHDS'
         41038 1997-1999/PY
L28           6 L16 NOT 1997-1999/PY

FILE 'BIOSIS'
       1680795 1997-1999/PY
L29         282 L17 NOT 1997-1999/PY

FILE 'EMBASE'
       1252969 1997-1999/PY
L30         207 L18 NOT 1997-1999/PY

FILE 'HCAPLUS'
       2535538 1997-1999/PY
L31         263 L19 NOT 1997-1999/PY

FILE 'NTIS'
         85373 1997-1999/PY
L32           7 L20 NOT 1997-1999/PY

FILE 'ESBIOBASE'
        831730 1997-1999/PY
L33         184 L21 NOT 1997-1999/PY

FILE 'BIOTECHNO'
        338670 1997-1999/PY
L34         143 L22 NOT 1997-1999/PY

FILE 'WPIDS'
       2351312 1997-1999/PY
L35          16 L23 NOT 1997-1999/PY

TOTAL FOR ALL FILES
L36        1795 L24 NOT 1997-1999/PY

=> s l36 not 2000-2003/py
FILE 'MEDLINE'
       1724843 2000-2003/PY
L37          23 L25 NOT 2000-2003/PY

FILE 'SCISEARCH'
       3248680 2000-2003/PY
L38          24 L26 NOT 2000-2003/PY

FILE 'LIFESCI'
        326324 2000-2003/PY
L39          11 L27 NOT 2000-2003/PY

FILE 'BIOTECHDS'
         60540 2000-2003/PY
L40           0 L28 NOT 2000-2003/PY

FILE 'BIOSIS'
       1778159 2000-2003/PY
L41          33 L29 NOT 2000-2003/PY
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FILE 'EMBASE'
       1483306 2000-2003/PY
L42          18 L30 NOT 2000-2003/PY

FILE 'HCAPLUS'
       3283625 2000-2003/PY
L43          21 L31 NOT 2000-2003/PY

FILE 'NTIS'
         53037 2000-2003/PY
L44           2 L32 NOT 2000-2003/PY

FILE 'ESBIOBASE'
        948733 2000-2003/PY
L45           0 L33 NOT 2000-2003/PY

FILE 'BIOTECHNO'
        388989 2000-2003/PY
L46          13 L34 NOT 2000-2003/PY

FILE 'WPIDS'
       2930742 2000-2003/PY
L47           0 L35 NOT 2000-2003/PY

TOTAL FOR ALL FILES
L48         145 L36 NOT 2000-2003/PY

=> dup rem l48
PROCESSING COMPLETED FOR L48
L49           46 DUP REM L48 (99 DUPLICATES REMOVED)

=> d tot

L49  ANSWER 1 OF 46     MEDLINE                          DUPLICATE 1
TI   The carboxyl-terminal transactivation domain of human serum response
     factor contains DNA-activated protein
     kinase phosphorylation sites.
SO   JOURNAL OF BIOLOGICAL CHEMISTRY, (1993 Oct 5) 268 (28) 21147-54.
     Journal code: 2985121R. ISSN: 0021-9258.
AU   Liu S H; Ma J T; Yueh A Y; Lees-Miller S P; Anderson C W; Ng S Y
AN   94012665     MEDLINE

L49  ANSWER 2 OF 46     MEDLINE
TI   Characterization of protein kinase activities associated with p53-large-T
     immune complexes from SV40-transformed rat cells.
SO   ONCOGENE, (1993 Aug) 8 (8) 2193-205.
     Journal code: 8711562. ISSN: 0950-9232.
AU   Muller E; Boldyreff B; Scheidtmann K H
AN   93330560     MEDLINE

L49  ANSWER 3 OF 46     MEDLINE                          DUPLICATE 2
TI   Mutation of the serine 15 phosphorylation site of human p53 reduces the
     ability of p53 to inhibit cell cycle progression.
SO   ONCOGENE, (1993 Jun) 8 (6) 1519-28.
     Journal code: 8711562. ISSN: 0950-9232.
AU   Fiscella M; Ullrich S J; Zambrano N; Shields M T; Lin D; Lees-Miller S P;
     Anderson C W; Mercer W E; Appella E
AN   93275646     MEDLINE

L49  ANSWER 4 OF 46     MEDLINE                          DUPLICATE 3
TI   c-Jun is phosphorylated by the DNA-dependent protein kinase in vitro;
     definition of the minimal kinase recognition motif.
SO   NUCLEIC ACIDS RESEARCH, (1993 Mar 11) 21 (5) 1289-95.
```

Journal code: 0411011. ISSN: 0305-1048.
AU Bannister A J; Gottlieb T M; Kouzarides T; Jackson S P
AN 93219094 MEDLINE

L49 ANSWER 5 OF 46 MEDLINE
TI Transcription factor phosphorylation by the DNA-dependent protein kinase.
SO BIOCHEMICAL SOCIETY TRANSACTIONS, (1993 Nov) 21 (4) 930-5. Ref: 38
Journal code: 7506897. ISSN: 0300-5127.
AU Finnie N; Gottlieb T; Hartley K; Jackson S P
AN 94178642 MEDLINE

L49 ANSWER 6 OF 46 MEDLINE DUPLICATE 4
TI Stimulation of phosphorylation of a nuclear protein (NP-34) in cultured Alzheimer's disease (AD) fibroblasts by interferon response element and other double-stranded oligonucleotides.
SO BIOCHEMISTRY AND MOLECULAR BIOLOGY INTERNATIONAL, (1993 Jul) 30 (4) 675-84.
Journal code: 9306673. ISSN: 1039-9712.
AU An S; Maturana J; Wu J M
AN 94004608 MEDLINE

L49 ANSWER 7 OF 46 MEDLINE DUPLICATE 5
TI DNA damage and the **DNA-activated protein kinase**.
SO TRENDS IN BIOCHEMICAL SCIENCES, (1993 Nov) 18 (11) 433-7. Ref: 32
Journal code: 7610674. ISSN: 0968-0004.
AU Anderson C W
AN 94120555 MEDLINE

L49 ANSWER 8 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI **DNA-ACTIVATED PROTEIN-KINASE** IN RAJI BURKITT-LYMPHOMA CELLS - PHOSPHORYLATION OF NUCLEAR-PROTEIN FACTORS INVOLVED IN DNA-REPLICATION AND TRANSCRIPTION
SO JOURNAL OF CELLULAR BIOCHEMISTRY, (09 JAN 1993) Supp. 17A, pp. 305. ISSN: 0730-2312.
AU TERAOKA H (Reprint); WATANABE F; MINAMI H; IIJIMA S; TSUKADA K; DATE T; KITAJIMA S
AN 93:124123 SCISEARCH

L49 ANSWER 9 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI **DNA-activated protein kinase** in Raji Burkitt's lymphoma cells: Phosphorylation of nuclear protein factors involved in DNA replication and transcription.
SO Journal of Cellular Biochemistry Supplement, (1993) Vol. 0, No. 17 PART A, pp. 305.
Meeting Info.: Keystone Symposium on Phosphorylation/Dephosphorylation in Signal Transduction Keystone, Colorado, USA January 17-24, 1993
ISSN: 0733-1959.
AU Teraoka, Hirobumi (1); Watanabe, Fumiaki (1); Minami, Hanae (1); Iijima, Shigeyuki (1); Tsukada, Kinji (1); Date, Takayasu; Kitajima, Shigetaka (1)
AN 1993:181735 BIOSIS

L49 ANSWER 10 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI ACTIVITY OF THE DNA-DEPENDENT PROTEIN-KINASE (**DNA-PK**) MAY REQUIRE 2 DISTINCT PROTEINS
SO JOURNAL OF CELLULAR BIOCHEMISTRY, (09 JAN 1993) Supp. 17A, pp. 304. ISSN: 0730-2312.
AU SUN S S (Reprint); MALIK N P; LUGOAYALA E; VANCUROVA I; CARTER T H
AN 93:124122 SCISEARCH

L49 ANSWER 11 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Activity of the DNA-dependent protein kinase (**DNA-PK**) may require two distinct proteins.
SO Journal of Cellular Biochemistry Supplement, (1993) Vol. 0, No. 17 PART A,

pp. 304.
Meeting Info.: Keystone Symposium on Phosphorylation/Dephosphorylation in Signal Transduction Keystone, Colorado, USA January 17-24, 1993
ISSN: 0733-1959.
AU Sun, Shi-Shin; Malik, Nusrat Parveen; Vancurova, Elissa Vv Lugo-Ayalaana; Carter, Timothy H.
AN 1993:181734 BIOSIS

L49 ANSWER 12 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 6
TI IDENTIFICATION AND CLONING OF DNA-SEQUENCES THAT BIND AND ACTIVATE THE DNA-DEPENDENT PROTEIN-KINASE (**DNA-PK**)
SO JOURNAL OF CELLULAR BIOCHEMISTRY, (09 JAN 1993) Supp. 17A, pp. 301.
ISSN: 0730-2312.
AU MALIK N P (Reprint); POLTORATSKY V; VANCUROVA I; CARTER T H
AN 93:124107 SCISEARCH

L49 ANSWER 13 OF 46 MEDLINE
TI Phosphorylation of transcription factor Sp1 by the DNA-dependent protein kinase.
SO ADVANCES IN SECOND MESSENGER AND PHOSPHOPROTEIN RESEARCH, (1993) 28 279-86. Ref: 27
Journal code: 8807408. ISSN: 1040-7952.
AU Jackson S; Gottlieb T; Hartley K
AN 94001138 MEDLINE

L49 ANSWER 14 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI THE KU (P70/P80) PROTEIN ASSEMBLES INTO AN AUTOIMMUNOGENIC COMPLEX WITH THE 350 KDA CATALYTIC POLYPEPTIDE OF DNA-DEPENDENT PROTEIN-KINASE (**DNA-PK**)
SO CLINICAL RESEARCH, (APR 1993) Vol. 41, No. 2, pp. A187.
ISSN: 0009-9279.
AU SUWA A (Reprint); HIRAKATA M; TAKEDA Y; MIMORI T; HARDIN J A
AN 93:227715 SCISEARCH

L49 ANSWER 15 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI The Ku (p70/p80) protein assembles into an autoimmunogenic complex with the 350 kDa catalytic polypeptide of DNA-dependent protein kinase (**DNA-PK**.
SO Clinical Research, (1993) Vol. 41, No. 2, pp. 187A.
Meeting Info.: Joint Meeting of the Association of American Physicians, the American Society for Clinical Investigation, and the American Federation for Clinical Research Washington, DC, USA April 30-May 3, 1993
ISSN: 0009-9279.
AU Suwa, A. (1); Hirakata, M.; Takeda, Y.; Mimori, T.; Hardin, J. A.
AN 1993:293076 BIOSIS

L49 ANSWER 16 OF 46 MEDLINE DUPLICATE 7
TI **DNA-activated protein kinase**.
SO TANPAKUSHITSU KAKUSAN KOSO. PROTEIN, NUCLEIC ACID, ENZYME, (1993 Feb) 38 (2) 158-65. Ref: 28
Journal code: 0413762. ISSN: 0039-9450.
AU Iijima S; Teraoka H
AN 93197559 MEDLINE

L49 ANSWER 17 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 8
TI PHOSPHORYLATION OF THE P53 TUMOR SUPPRESSOR PROTEIN BY THE **DNA-ACTIVATED PROTEIN-KINASE**
SO JOURNAL OF CELLULAR BIOCHEMISTRY, (13 MAR 1993) Supp. 17D, pp. 157.
ISSN: 0730-2312.
AU ANDERSON C W (Reprint); LEESMILLER S P; LIN D; MERCER W E; SAKAGUCHI K; FISCELLA M; ULLRICH S; APPELLA E
AN 93:220194 SCISEARCH

L49 ANSWER 18 OF 46 MEDLINE DUPLICATE 9

TI The DNA-dependent protein kinase: requirement for DNA ends and association with Ku antigen.
SO CELL, (1993 Jan 15) 72 (1) 131-42.
Journal code: 0413066. ISSN: 0092-8674.
AU Gottlieb T M; Jackson S P
AN 93137321 MEDLINE

L49 ANSWER 19 OF 46 MEDLINE DUPLICATE 10
TI Phosphorylation of a 72-kDa nucleoprotein (NP-72) in HL-60 cells is mediated by the double-stranded DNA-dependent protein kinase (**DNA -PK**).
SO BIOCHEMISTRY AND MOLECULAR BIOLOGY INTERNATIONAL, (1993 Sep) 31 (1) 113-24.
Journal code: 9306673. ISSN: 1039-9712.
AU Konno-Sato S; Wu J M; Carter T H
AN 94083934 MEDLINE

L49 ANSWER 20 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI BINDING OF KU-DEPENDENT AND DNA-DEPENDENT PROTEIN-KINASE (**DNA-PK**) TO A NOVEL CHROMOSOMAL RECEPTOR
SO ARTHRITIS AND RHEUMATISM, (SEP 1993) Vol. 36, No. 9, Supp. S, pp. S72. ISSN: 0004-3591.
AU SATOH M (Reprint); AJMANI A K; REEVES W H
AN 93:639432 SCISEARCH

L49 ANSWER 21 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Binding of ku and DNA-dependent protein kinase (**DNA-PK**) to a novel chromosomal receptor.
SO Arthritis and Rheumatism, (1993) Vol. 36, No. 9 SUPPL., pp. S72.
Meeting Info.: 57th Annual Scientific Meeting of the American College of Rheumatology San Antonio, Texas, USA November 7-11, 1993
ISSN: 0004-3591.
AU Satoh, Minoru; Ajmani, Ajay K.; Reeves, Westley H.
AN 1994:9088 BIOSIS

L49 ANSWER 22 OF 46 MEDLINE DUPLICATE 11
TI Murine monoclonal antibodies specific for conserved and non-conserved antigenic determinants of the human and murine Ku autoantigens.
SO MOLECULAR BIOLOGY REPORTS, (1993 Jun) 18 (1) 15-28.
Journal code: 0403234. ISSN: 0301-4851.
AU Wang J; Chou C H; Blankson J; Satoh M; Knuth M W; Eisenberg R A; Pisetsky D S; Reeves W H
AN 94049782 MEDLINE

L49 ANSWER 23 OF 46 MEDLINE DUPLICATE 12
TI Phosphorylation of protein tau by double-stranded DNA-dependent protein kinase.
SO BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1993 May 28) 193 (1) 13-8.
Journal code: 0372516. ISSN: 0006-291X.
AU Wu J M; Chen Y; An S; Perruccio L; Abdel-Ghany M; Carter T H
AN 93277540 MEDLINE

L49 ANSWER 24 OF 46 MEDLINE DUPLICATE 13
TI Human **DNA-activated protein kinase** phosphorylates serines 15 and 37 in the amino-terminal transactivation domain of human p53.
SO MOLECULAR AND CELLULAR BIOLOGY, (1992 Nov) 12 (11) 5041-9.
Journal code: 8109087. ISSN: 0270-7306.
AU Lees-Miller S P; Sakaguchi K; Ullrich S J; Appella E; Anderson C W
AN 93024450 MEDLINE

L49 ANSWER 25 OF 46 MEDLINE DUPLICATE 14
TI Phosphorylation sites in the amino-terminal region of mouse p53.

SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1992 May 15) 89 (10) 4231-5.
Journal code: 7505876. ISSN: 0027-8424.
AU Wang Y; Eckhart W
AN 92262414 MEDLINE

L49 ANSWER 26 OF 46 MEDLINE DUPLICATE 15
TI **DNA-activated protein kinase** in Raji Burkitt's lymphoma cells. Phosphorylation of c-Myc oncoprotein.
SO EUROPEAN JOURNAL OF BIOCHEMISTRY, (1992 Jun 1) 206 (2) 595-603.
Journal code: 0107600. ISSN: 0014-2956.
AU Iijima S; Teraoka H; Date T; Tsukada K
AN 92283288 MEDLINE

L49 ANSWER 27 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI SURFACE-ENHANCED RAMAN-SPECTROSCOPY OF ELLIPTICINE, 2-N-METHYLELLIPTICINIUM AND THEIR COMPLEXES WITH DNA
SO JOURNAL OF RAMAN SPECTROSCOPY, (JUL 1992) Vol. 23, No. 7, pp. 373-377.
ISSN: 0377-0486.
AU AUBARD J (Reprint); SCHWALLER M A; PANTIGNY J; MARSAULT J P; LEVI G
AN 92:447455 SCISEARCH

L49 ANSWER 28 OF 46 MEDLINE DUPLICATE 16
TI The nuclear serine/threonine protein kinase **DNA-PK**.
SO CRITICAL REVIEWS IN EUKARYOTIC GENE EXPRESSION, (1992) 2 (4) 283-314.
Ref: 187
Journal code: 9007261. ISSN: 1045-4403.
AU Anderson C W; Lees-Miller S P
AN 93136568 MEDLINE

L49 ANSWER 29 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI HUMAN SERUM RESPONSE FACTOR SRF IS PHOSPHORYLATED BY THE **DNA-ACTIVATED PROTEIN KINASE DNA-PK**.
SO KEYSTONE SYMPOSIUM ON POSITIVE GROWTH CONTROL, KEYSTONE, COLORADO, USA, JANUARY 26-FEBRUARY 2, 1992. J CELL BIOCHEM SUPPL. (1992) 0 (16 PART B), 242.
CODEN: JCBSD7.
AU NG S-Y; LIU S-H; MA J-T; CHIANG S-H; LEES-MILLER S P; ANDERSON C W
AN 1992:293572 BIOSIS

L49 ANSWER 30 OF 46 LIFESCI COPYRIGHT 2003 CSA
TI The DNA-dependent protein kinase: Requirement for DNA ends and association with Ku antigen.
SO CELL., (1992) vol. 72, no. 1, pp. 131-142.
AU Gottlieb, T.M.; Jackson, S.P.
AN 93:64479 LIFESCI

L49 ANSWER 31 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 17
TI STIMULATION OF PHOSPHORYLATION OF A NUCLEAR-PROTEIN (NP-72) IN CONTROL AND TPA TREATED HL-60 CELLS BY VARIOUS DSDNA OLIGONUCLEOTIDES IN THE PRESENCE OF DSDNA DEPENDENT PROTEIN-KINASE (**DNA-PK**)
SO FASEB JOURNAL, (01 JAN 1992) Vol. 6, No. 1, pp. A69.
ISSN: 0892-6638.
AU YESUS K G (Reprint); MATURANA J A; KONNO S; CARTER T H; WU J M
AN 92:39334 SCISEARCH

L49 ANSWER 32 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI STIMULATION OF PHOSPHORYLATION OF A NUCLEAR PROTEIN NP-72 IN CONTROL AND TPA TREATED HL-60 CELLS BY VARIOUS DSDNA OLIGONUCLEOTIDES IN THE PRESENCE OF DSDNA DEPENDENT PROTEIN KINASE **DNA-PK**.
SO JOINT ANNUAL MEETING OF THE BIOPHYSICAL SOCIETY AND THE AMERICAN SOCIETY FOR BIOCHEMISTRY AND MOLECULAR BIOLOGY, HOUSTON, TEXAS, USA, FEBRUARY 9-13, 1992. BIOPHYS J. (1992) 61 (2 PART 2), A69.

CODEN: BIOJAU. ISSN: 0006-3495.
AU YESUS K G; MATURANA J A; KONNO S; CARTER T H; WU J M
AN 1992:201601 BIOSIS

L49 ANSWER 33 OF 46 MEDLINE DUPLICATE 18
TI Chicken progesterone receptor is phosphorylated by a DNA-dependent protein kinase during in vitro transcription assays.
SO MOLECULAR ENDOCRINOLOGY, (1992 Jan) 6 (1) 8-14.
Journal code: 8801431. ISSN: 0888-8809.
AU Weigel N L; Carter T H; Schrader W T; O'Malley B W
AN 92149580 MEDLINE

L49 ANSWER 34 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI PROGRESS IN MOLECULAR AND SUBCELLULAR BIOLOGY VOL. 12.
SO JEANTEUR, P., ET AL. (ED.). PROGRESS IN MOLECULAR AND SUBCELLULAR BIOLOGY, VOL. 12. IX+137P. SPRINGER-VERLAG: BERLIN, GERMANY; NEW YORK, NEW YORK, USA. ILLUS. (1991) 0 (0), IX+137P.
CODEN: PMSBA4. ISSN: 0079-6484. ISBN: 3-540-53900-X, 0-387-53900-X.
AU JEANTEUR P; ET AL
AN 1992:378112 BIOSIS

L49 ANSWER 35 OF 46 MEDLINE DUPLICATE 19
TI The human **DNA-activated protein kinase** phosphorylates simian virus 40 T antigen at amino- and carboxy-terminal sites.
SO JOURNAL OF VIROLOGY, (1991 Oct) 65 (10) 5131-40.
Journal code: 0113724. ISSN: 0022-538X.
AU Chen Y R; Lees-Miller S P; Tegtmeyer P; Anderson C W
AN 91374560 MEDLINE

L49 ANSWER 36 OF 46 MEDLINE DUPLICATE 20
TI The **DNA-activated protein kinase, DNA-PK**: a potential coordinator of nuclear events.
SO CANCER CELLS, (1991 Sep) 3 (9) 341-6. Ref: 34
Journal code: 9000382. ISSN: 1042-2196.
AU Lees-Miller S P; Anderson C W
AN 92088810 MEDLINE

L49 ANSWER 37 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI MEASUREMENT OF **DNA-ACTIVATED PROTEIN KINASE** ABUNDANCE SYNTHESIS AND ACTIVITY DURING THE CELL-CYCLE.
SO ABSTRACTS OF PAPERS PRESENTED AT THE THIRTY-FIRST ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CELL BIOLOGY, BOSTON, MASSACHUSETTS, USA, DECEMBER 8-12, 1991. J CELL BIOL. (1991) 115 (3 PART 2), 283A.
CODEN: JCLBA3. ISSN: 0021-9525.
AU SUN I; CARTER T H
AN 1992:88367 BIOSIS

L49 ANSWER 38 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 21
TI THE **DNA-ACTIVATED PROTEIN KINASE DNA-PK**.
SO JEANTEUR, P., ET AL. (ED.). PROGRESS IN MOLECULAR AND SUBCELLULAR BIOLOGY, VOL. 12. IX+137P. SPRINGER-VERLAG: BERLIN, GERMANY; NEW YORK, NEW YORK, USA. ILLUS. (1991) 0 (0), 37-57.
CODEN: PMSBA4. ISSN: 0079-6484. ISBN: 3-540-53900-X, 0-387-53900-X.
AU CARTER T H; ANDERSON C W
AN 1992:378114 BIOSIS

L49 ANSWER 39 OF 46 MEDLINE DUPLICATE 22
TI Human cells contain a **DNA-activated protein kinase** that phosphorylates simian virus 40 T antigen, mouse p53, and the human Ku autoantigen.
SO MOLECULAR AND CELLULAR BIOLOGY, (1990 Dec) 10 (12) 6472-81.

Journal code: 8109087. ISSN: 0270-7306.
AU Lees-Miller S P; Chen Y R; Anderson C W
AN 91061755 MEDLINE

L49 ANSWER 40 OF 46 MEDLINE DUPLICATE 23
TI A **DNA-activated protein kinase** from HeLa cell nuclei.
SO MOLECULAR AND CELLULAR BIOLOGY, (1990 Dec) 10 (12) 6460-71.
Journal code: 8109087. ISSN: 0270-7306.
AU Carter T; Vancurova I; Sun I; Lou W; DeLeon S
AN 91061754 MEDLINE

L49 ANSWER 41 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISI
TI A **DNA-ACTIVATED PROTEIN-KINASE** FROM HELA-CELL NUCLEI
SO MOLECULAR AND CELLULAR BIOLOGY, (1990) Vol. 10, No. 12, pp. 6460-6471.
AU CARTER T (Reprint); VANCUROVA I; SUN I; LOU W; DELEON S
AN 90:633085 SCISEARCH

L49 ANSWER 42 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI SV-40 LARGE TUMOR ANTIGEN AND THE TUMOR SUPPRESSOR P53 ARE PHOSPHORYLATED BY A **DNA-ACTIVATED PROTEIN KINASE** FROM HUMAN CELLS.
SO EIGHTH INTERNATIONAL CONFERENCE ON METHODS IN PROTEIN SEQUENCE ANALYSIS, KIRUNA, SWEDEN, JULY 1-6, 1990. J PROTEIN CHEM. (1990) 9 (3), 306-307.
CODEN: JPCHD2. ISSN: 0277-8033.
AU LEES-MILLER S P; CHEN Y-R; ANDERSON C W
AN 1990:484135 BIOSIS

L49 ANSWER 43 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI INTERACTIONS OF THE HUMAN **DNA-ACTIVATED PROTEIN KINASE** PKD WITH SYNTHETIC ENHANCER OLIGONUCLEOTIDES.
SO THIRTIETH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR CELL BIOLOGY, SAN DIEGO, CALIFORNIA, USA, DECEMBER 9-13, 1990. J CELL BIOL. (1990) 111 (5 PART 2), 134A.
CODEN: JCLBA3. ISSN: 0021-9525.
AU VANCUROVA I; CARTER T H
AN 1991:43905 BIOSIS

L49 ANSWER 44 OF 46 MEDLINE DUPLICATE 24
TI The human double-stranded **DNA-activated protein kinase** phosphorylates the 90-kDa heat-shock protein, hsp90 alpha at two NH2-terminal threonine residues.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1989 Oct 15) 264 (29) 17275-80.
Journal code: 2985121R. ISSN: 0021-9258.
AU Lees-Miller S P; Anderson C W
AN 90008887 MEDLINE

L49 ANSWER 45 OF 46 NTIS COPYRIGHT 2003 NTIS
TI PU 3-Kinase: A Pivotal Pathway in T-Cell Activation.
Reprint: PU 3-Kinase: A Pivotal Pathway in T-Cell Activation.
NR AD-A307 005/9/XAB; NMRI-96-04
13p; 1996
AU Ward, S. G.; June, C. H.; Olive, D.
AN 1996(19):06678 NTIS

L49 ANSWER 46 OF 46 NTIS COPYRIGHT 2003 NTIS
TI SV40 large tumor antigen and the tumor suppressor p53 are phosphorylated by a **DNA-activated protein kinase** from human cells.
NR DE90016556/XAB; BNL-43799, BIO-4569; CONF-9007160-1
4p; 1990
AU Lees-Miller, S. P.; Chen, Y. R.; Anderson, C. W.

```
AN     1991(13):02854   NTIS


=> s p53
FILE 'MEDLINE'
L50       28098 P53

FILE 'SCISEARCH'
L51       35120 P53

FILE 'LIFESCI'
L52        9520 P53

FILE 'BIOTECHDS'
L53        1014 P53

FILE 'BIOSIS'
L54       33991 P53

FILE 'EMBASE'
L55       26089 P53

FILE 'HCAPLUS'
L56       23690 P53

FILE 'NTIS'
L57         400 P53

FILE 'ESBIOBASE'
L58       16719 P53

FILE 'BIOTECHNO'
L59       14698 P53

FILE 'WPIDS'
L60        1006 P53

TOTAL FOR ALL FILES
L61      190345 P53

=> s l61(8a)(fragment# or peptide# or portion#)
FILE 'MEDLINE'
         202686 FRAGMENT#
         328016 PEPTIDE#
          85665 PORTION#
L62         519 L50(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'SCISEARCH'
         153564 FRAGMENT#
         238184 PEPTIDE#
          72679 PORTION#
L63         568 L51(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'LIFESCI'
          73702 FRAGMENT#
          87272 PEPTIDE#
          27722 PORTION#
L64         325 L52(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'BIOTECHDS'
          38001 FRAGMENT#
          23759 PEPTIDE#
           8518 PORTION#
L65          75 L53(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)
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FILE 'BIOSIS'
        179186 FRAGMENT#
        287886 PEPTIDE#
        101537 PORTION#
L66        612 L54(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'EMBASE'
        138512 FRAGMENT#
        210658 PEPTIDE#
         78707 PORTION#
L67        558 L55(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'HCAPLUS'
        281923 FRAGMENT#
        372530 PEPTIDE#
        281505 PORTION#
L68        793 L56(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'NTIS'
          7808 FRAGMENT#
          3667 PEPTIDE#
         32486 PORTION#
L69         10 L57(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'ESBIOBASE'
         54228 FRAGMENT#
         89705 PEPTIDE#
         24445 PORTION#
L70        394 L58(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'BIOTECHNO'
         95026 FRAGMENT#
        100719 PEPTIDE#
         24349 PORTION#
L71        456 L59(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

FILE 'WPIDS'
         43718 FRAGMENT#
         37856 PEPTIDE#
        950648 PORTION#
L72         93 L60(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

TOTAL FOR ALL FILES
L73       4403 L61(8A)(FRAGMENT# OR PEPTIDE# OR PORTION#)

=> s l73 not 2000-2003/py
FILE 'MEDLINE'
       1724843 2000-2003/PY
L74        361 L62 NOT 2000-2003/PY

FILE 'SCISEARCH'
       3248680 2000-2003/PY
L75        391 L63 NOT 2000-2003/PY

FILE 'LIFESCI'
        326324 2000-2003/PY
L76        241 L64 NOT 2000-2003/PY

FILE 'BIOTECHDS'
         60540 2000-2003/PY
L77         44 L65 NOT 2000-2003/PY

FILE 'BIOSIS'
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       1778159 2000-2003/PY
L78         423 L66 NOT 2000-2003/PY

FILE 'EMBASE'
       1483306 2000-2003/PY
L79         399 L67 NOT 2000-2003/PY

FILE 'HCAPLUS'
       3283625 2000-2003/PY
L80         480 L68 NOT 2000-2003/PY

FILE 'NTIS'
         53037 2000-2003/PY
L81           7 L69 NOT 2000-2003/PY

FILE 'ESBIOBASE'
        948733 2000-2003/PY
L82         255 L70 NOT 2000-2003/PY

FILE 'BIOTECHNO'
        388989 2000-2003/PY
L83         338 L71 NOT 2000-2003/PY

FILE 'WPIDS'
       2930742 2000-2003/PY
L84          24 L72 NOT 2000-2003/PY

TOTAL FOR ALL FILES
L85        2963 L73 NOT 2000-2003/PY

=> s l85 not 1997-1999/py
FILE 'MEDLINE'
       1333570 1997-1999/PY
L86         202 L74 NOT 1997-1999/PY

FILE 'SCISEARCH'
       2862496 1997-1999/PY
L87         210 L75 NOT 1997-1999/PY

FILE 'LIFESCI'
        337226 1997-1999/PY
L88         138 L76 NOT 1997-1999/PY

FILE 'BIOTECHDS'
         41038 1997-1999/PY
L89          24 L77 NOT 1997-1999/PY

FILE 'BIOSIS'
       1680795 1997-1999/PY
L90         248 L78 NOT 1997-1999/PY

FILE 'EMBASE'
       1252969 1997-1999/PY
L91         231 L79 NOT 1997-1999/PY

FILE 'HCAPLUS'
       2535538 1997-1999/PY
L92         261 L80 NOT 1997-1999/PY

FILE 'NTIS'
         85373 1997-1999/PY
L93           3 L81 NOT 1997-1999/PY

FILE 'ESBIOBASE'
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        831730 1997-1999/PY
L94        107 L82 NOT 1997-1999/PY

FILE 'BIOTECHNO'
        338670 1997-1999/PY
L95        206 L83 NOT 1997-1999/PY

FILE 'WPIDS'
       2351312 1997-1999/PY
L96          3 L84 NOT 1997-1999/PY

TOTAL FOR ALL FILES
L97       1633 L85 NOT 1997-1999/PY

=>
=> s l97 not 1994-1996/py
FILE 'MEDLINE'
       1244236 1994-1996/PY
L98         81 L86 NOT 1994-1996/PY

FILE 'SCISEARCH'
       2559592 1994-1996/PY
L99         59 L87 NOT 1994-1996/PY

FILE 'LIFESCI'
        325401 1994-1996/PY
L100        53 L88 NOT 1994-1996/PY

FILE 'BIOTECHDS'
         46905 1994-1996/PY
L101         7 L89 NOT 1994-1996/PY

FILE 'BIOSIS'
       1660749 1994-1996/PY
L102        95 L90 NOT 1994-1996/PY

FILE 'EMBASE'
       1117373 1994-1996/PY
L103        85 L91 NOT 1994-1996/PY

FILE 'HCAPLUS'
       2256014 1994-1996/PY
L104       114 L92 NOT 1994-1996/PY

FILE 'NTIS'
        131755 1994-1996/PY
L105         1 L93 NOT 1994-1996/PY

FILE 'ESBIOBASE'
        498223 1994-1996/PY
L106         3 L94 NOT 1994-1996/PY

FILE 'BIOTECHNO'
        304425 1994-1996/PY
L107        81 L95 NOT 1994-1996/PY

FILE 'WPIDS'
       1732428 1994-1996/PY
L108         0 L96 NOT 1994-1996/PY

TOTAL FOR ALL FILES
L109       579 L97 NOT 1994-1996/PY

=> s l109 and human
```

```
FILE 'MEDLINE'
       8110736 HUMAN
L110         51 L98 AND HUMAN

FILE 'SCISEARCH'
       1027166 HUMAN
L111         38 L99 AND HUMAN

FILE 'LIFESCI'
        318724 HUMAN
L112         24 L100 AND HUMAN

FILE 'BIOTECHDS'
         54727 HUMAN
L113          4 L101 AND HUMAN

FILE 'BIOSIS'
       5448376 HUMAN
L114         63 L102 AND HUMAN

FILE 'EMBASE'
       4707523 HUMAN
L115         59 L103 AND HUMAN

FILE 'HCAPLUS'
       1135506 HUMAN
L116         74 L104 AND HUMAN

FILE 'NTIS'
         80440 HUMAN
L117          0 L105 AND HUMAN

FILE 'ESBIOBASE'
        350395 HUMAN
L118          2 L106 AND HUMAN

FILE 'BIOTECHNO'
        684986 HUMAN
L119         58 L107 AND HUMAN

FILE 'WPIDS'
        123088 HUMAN
L120          0 L108 AND HUMAN

TOTAL FOR ALL FILES
L121        373 L109 AND HUMAN

=> dup rem l121
PROCESSING COMPLETED FOR L121
L122         115 DUP REM L121 (258 DUPLICATES REMOVED)

=> d tot

L122  ANSWER 1 OF 115  BIOTECHDS COPYRIGHT 2003 THOMSON DERWENT AND ISI
TI    Human protein p53 binding to major histocompatibility complex
      class I molecule;
         cytotoxic T-lymphocyte response in cell culture for application in
         cancer diagnosis and therapy
AN    1994-01949  BIOTECHDS
PI    WO 9324525 9 Dec 1993

L122 ANSWER 2 OF 115     MEDLINE                          DUPLICATE 1
TI   Analysis of p53 antibodies in patients with various cancers define B-cell
     epitopes of human p53: distribution on primary structure and
```

exposure on protein surface.
SO CANCER RESEARCH, (1993 Dec 15) 53 (24) 5872-6.
Journal code: 2984705R. ISSN: 0008-5472.
AU Lubin R; Schlichtholz B; Bengoufa D; Zalcman G; Tredaniel J; Hirsch A; de Fromentel C C; Preudhomme C; Fenaux P; Fournier G; +
AN 94084640 MEDLINE

L122 ANSWER 3 OF 115 MEDLINE DUPLICATE 2
TI Absence of p53 gene mutations in primary neuroblastomas.
SO CANCER RESEARCH, (1993 Nov 1) 53 (21) 5269-73.
Journal code: 2984705R. ISSN: 0008-5472.
AU Vogan K; Bernstein M; Leclerc J M; Brisson L; Brossard J; Brodeur G M; Pelletier J; Gros P
AN 94036809 MEDLINE

L122 ANSWER 4 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Redox modulation of p53 conformation and sequence-specific DNA binding in vitro.
SO Cancer Research, (1993) Vol. 53, No. 19, pp. 4469-4473.
ISSN: 0008-5472.
AU Hainaut, Pierre (1); Milner, Jo
AN 1993:525110 BIOSIS

L122 ANSWER 5 OF 115 MEDLINE DUPLICATE 3
TI Mapping of the p53 and mdm-2 interaction domains.
SO MOLECULAR AND CELLULAR BIOLOGY, (1993 Jul) 13 (7) 4107-14.
Journal code: 8109087. ISSN: 0270-7306.
AU Chen J; Marechal V; Levine A J
AN 93309444 MEDLINE

L122 ANSWER 6 OF 115 MEDLINE DUPLICATE 4
TI 'Cold SSCP': a simple, rapid and non-radioactive method for optimized single-strand conformation polymorphism analyses.
SO NUCLEIC ACIDS RESEARCH, (1993 Aug 11) 21 (16) 3637-42.
Journal code: 0411011. ISSN: 0305-1048.
AU Hongyo T; Buzard G S; Calvert R J; Weghorst C M
AN 93376481 MEDLINE

L122 ANSWER 7 OF 115 MEDLINE DUPLICATE 5
TI A mutant **p53** tumor suppressor protein is a target for **peptide**-induced CD8+ cytotoxic T-cells.
SO CANCER RESEARCH, (1993 Jul 15) 53 (14) 3257-61.
Journal code: 2984705R. ISSN: 0008-5472.
AU Yanuck M; Carbone D P; Pendleton C D; Tsukui T; Winter S F; Minna J D; Berzofsky J A
AN 93313855 MEDLINE

L122 ANSWER 8 OF 115 HCAPLUS COPYRIGHT 2003 ACS
TI Sequence-specific DNA binding by p53: identification of target sites and lack of binding to p53-MDM2 complexes
SO EMBO Journal (1993), 12(7), 2799-808
CODEN: EMJODG; ISSN: 0261-4189
AU Zauberman, Arie; Barak, Yaacov; Ragimov, Naomi; Levy, Naomi; Oren, Moshe
AN 1993:488338 HCAPLUS
DN 119:88338

L122 ANSWER 9 OF 115 MEDLINE DUPLICATE 6
TI A proteolytic **fragment** from the central region of **p53** has marked sequence-specific DNA-binding activity when generated from wild-type but not from oncogenic mutant p53 protein.
SO GENES AND DEVELOPMENT, (1993 Dec) 7 (12B) 2565-74.
Journal code: 8711660. ISSN: 0890-9369.
AU Bargonetti J; Manfredi J J; Chen X; Marshak D R; Prives C
AN 94102538 MEDLINE

L122 ANSWER 10 OF 115 MEDLINE DUPLICATE 7
TI The DNA-binding domain of p53 contains the four conserved regions and the major mutation hot spots.
SO GENES AND DEVELOPMENT, (1993 Dec) 7 (12B) 2556-64.
Journal code: 8711660. ISSN: 0890-9369.
AU Pavletich N P; Chambers K A; Pabo C O
AN 94102537 MEDLINE

L122 ANSWER 11 OF 115 LIFESCI COPYRIGHT 2003 CSA
TI The DNA-binding domain of p53 contains the four conserved regions and the major mutation hot spots
SO GENES DEV., (1993) vol. 7, no. 126, pp. 2556-2564.
ISSN: 0890-9369.
AU Pavletich, N.P.; Chambers, K.A.; Pabo, C.O.
AN 94:27791 LIFESCI

L122 ANSWER 12 OF 115 MEDLINE DUPLICATE 8
TI Direct interaction between the transcriptional activation domain of **human** p53 and the TATA box-binding protein.
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1993 Feb 5) 268 (4) 2284-7.
Journal code: 2985121R. ISSN: 0021-9258.
AU Truant R; Xiao H; Ingles C J; Greenblatt J
AN 93155022 MEDLINE

L122 ANSWER 13 OF 115 MEDLINE DUPLICATE 9
TI An HLA-A11-specific motif in nonamer peptides derived from viral and cellular proteins.
SO PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (1993 Mar 15) 90 (6) 2217-21.
Journal code: 7505876. ISSN: 0027-8424.
AU Zhang Q J; Gavioli R; Klein G; Masucci M G
AN 93211933 MEDLINE

L122 ANSWER 14 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
DUPLICATE 10
TI "Hot spots" mutation analysis of p53 gene in gastrointestinal cancers by amplification of naturally occurring and artificially created restriction sites.
SO Clinical Chemistry, (1993) Vol. 39, No. 10, pp. 2186-2191.
ISSN: 0009-9147.
AU Chen, Pao-Huei (1); Lin, Shyr-Yi; Wang, Chung-Kwe; Chen, Yi-Jen; Chen, Te-Chuan; Chang, Jan-Gowth
AN 1993:585486 BIOSIS

L122 ANSWER 15 OF 115 MEDLINE DUPLICATE 11
TI Heterogeneity of transcriptional activity of mutant p53 proteins and p53 DNA target sequences.
SO ONCOGENE, (1993 Aug) 8 (8) 2159-66.
Journal code: 8711562. ISSN: 0950-9232.
AU Chen J Y; Funk W D; Wright W E; Shay J W; Minna J D
AN 93330556 MEDLINE

L122 ANSWER 16 OF 115 MEDLINE DUPLICATE 12
TI In vitro induction of **human** cytotoxic T lymphocyte responses against **peptides** of mutant and wild-type **p53**.
SO EUROPEAN JOURNAL OF IMMUNOLOGY, (1993 Sep) 23 (9) 2072-7.
Journal code: 1273201. ISSN: 0014-2980.
AU Houbiers J G; Nijman H W; van der Burg S H; Drijfhout J W; Kenemans P; van de Velde C J; Brand A; Momburg F; Kast W M; Melief C J
AN 93380471 MEDLINE

L122 ANSWER 17 OF 115 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 13
TI ALTERATIONS OF THE P53 TUMOR SUPPRESSOR GENE AND ITS ASSOCIATION WITH

ACTIVATION OF THE C-K-RAS-2 PROTOONCOGENE IN PREMALIGNANT AND MALIGNANT LESIONS OF THE **HUMAN** UTERINE ENDOMETRIUM
SO CANCER RESEARCH, (15 APR 1993) Vol. 53, No. 8, pp. 1883-1888.
ISSN: 0008-5472.
AU ENOMOTO T (Reprint); FUJITA M; INOUE M; RICE J M; NAKAJIMA R; TANIZAWA O; NOMURA T
AN 93:247083 SCISEARCH

L122 ANSWER 18 OF 115 MEDLINE
TI Cooperative DNA binding of p53 with TFIID (TBP): a possible mechanism for transcriptional activation.
SO GENES AND DEVELOPMENT, (1993 Oct) 7 (10) 1837-49.
Journal code: 8711660. ISSN: 0890-9369.
AU Chen X; Farmer G; Zhu H; Prywes R; Prives C
AN 94010283 MEDLINE

L122 ANSWER 19 OF 115 MEDLINE DUPLICATE 14
TI Use of the two-hybrid system to identify the domain of p53 involved in oligomerization.
SO ONCOGENE, (1993 Jun) 8 (6) 1693-6.
Journal code: 8711562. ISSN: 0950-9232.
AU Iwabuchi K; Li B; Bartel P; Fields S
AN 93275666 MEDLINE

L122 ANSWER 20 OF 115 MEDLINE DUPLICATE 15
TI Pancreatic adenocarcinomas frequently show p53 gene mutations.
SO AMERICAN JOURNAL OF PATHOLOGY, (1993 May) 142 (5) 1534-43.
Journal code: 0370502. ISSN: 0002-9440.
AU Scarpa A; Capelli P; Mukai K; Zamboni G; Oda T; Iacono C; Hirohashi S
AN 93263220 MEDLINE

L122 ANSWER 21 OF 115 MEDLINE DUPLICATE 16
TI State of p53, Rb and DCC tumor suppressor genes in **human** oral cancer cell lines.
SO ANTICANCER RESEARCH, (1993 Sep-Oct) 13 (5A) 1405-13.
Journal code: 8102988. ISSN: 0250-7005.
AU Kim M S; Li S L; Bertolami C N; Cherrick H M; Park N H
AN 94058082 MEDLINE

L122 ANSWER 22 OF 115 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 17
TI HIGH-LEVELS OF P53 PROTEIN EXPRESSION DO NOT CORRELATE WITH P53 GENE-MUTATIONS IN ANAPLASTIC LARGE-CELL LYMPHOMA
SO AMERICAN JOURNAL OF PATHOLOGY, (SEP 1993) Vol. 143, No. 3, pp. 845-856.
ISSN: 0002-9440.
AU CESARMAN E; INGHIRAMI G; CHADBURN A; KNOWLES D M (Reprint)
AN 93:570858 SCISEARCH

L122 ANSWER 23 OF 115 HCAPLUS COPYRIGHT 2003 ACS
TI WAF1, a potential mediator of p53 tumor suppression
SO Cell (Cambridge, MA, United States) (1993), 75(4), 817-25
CODEN: CELLB5; ISSN: 0092-8674
AU El-Deiry, Wafik S.; Tokino, Takashi; Velculescu, Victor E.; Levy, Daniel B.; Parsons, Ramon; Trent, Jeffrey M.; Lin, David; Mercer, W. Edward; Kinzler, Kenneth W.; Vogelstein, Bert
AN 1994:97539 HCAPLUS
DN 120:97539

L122 ANSWER 24 OF 115 HCAPLUS COPYRIGHT 2003 ACS
TI Release of the p53-induced repression on thymidine kinase promoter by single p53-binding sequence
SO Biochemical and Biophysical Research Communications (1993), 191(2), 662-8
CODEN: BBRCA9; ISSN: 0006-291X
AU Yuan, Jean Nian; Liu, Biing Hui; Lee, Heng; Shaw, Yueh Tsern; Chiou, Shean Tai; Chang, Wen Chang; Lai, Ming Derg

AN 1993:227382 HCAPLUS
DN 118:227382

L122 ANSWER 25 OF 115 MEDLINE DUPLICATE 18
TI Lack of allelic deletion and point mutation as mechanisms of p53 activation in **human** malignant melanoma.
SO INTERNATIONAL JOURNAL OF CANCER, (1993 Oct 21) 55 (4) 562-5.
Journal code: 0042124. ISSN: 0020-7136.
AU Castresana J S; Rubio M P; Vazquez J J; Idoate M; Sober A J; Seizinger B R; Barnhill R L
AN 94011506 MEDLINE

L122 ANSWER 26 OF 115 MEDLINE DUPLICATE 19
TI Current topics of immunohistochemistry as applied to skin tumors.
SO JOURNAL OF DERMATOLOGY, (1993 Sep) 20 (9) 521-32. Ref: 61
Journal code: 7600545. ISSN: 0385-2407.
AU Hashimoto K; Fujiwara K; Mehregan A
AN 94044149 MEDLINE

L122 ANSWER 27 OF 115 MEDLINE DUPLICATE 20
TI Molecular genetics of pediatric brain stem gliomas. Application of PCR techniques to small and archival brain tumor specimens.
SO JOURNAL OF NEUROPATHOLOGY AND EXPERIMENTAL NEUROLOGY, (1993 Sep) 52 (5) 507-15.
Journal code: 2985192R. ISSN: 0022-3069.
AU Louis D N; Rubio M P; Correa K M; Gusella J F; von Deimling A
AN 93367522 MEDLINE

L122 ANSWER 28 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
TI Endogenous **p53** tumor suppressor protein is a target for **peptide**-induced CD8 positive cytotoxic T cells.
SO Proceedings of the American Association for Cancer Research Annual Meeting, (1993) Vol. 34, No. 0, pp. 491.
Meeting Info.: 84th Annual Meeting of the American Association for Cancer Research Orlando, Florida, USA May 19-22, 1993
ISSN: 0197-016X.
AU Carbone, David P. (1); Yanuck, Michael; Pendleton, C. David; Tsukui, Taku; Winter, Stefan; Minna, John D. (1); Berzofsky, Jay A.
AN 1993:400810 BIOSIS

L122 ANSWER 29 OF 115 MEDLINE DUPLICATE 21
TI p53 gene mutations and 17p allelic deletions in hepatocellular carcinoma from Japan.
SO CANCER, (1993 Jul 15) 72 (2) 355-60.
Journal code: 0374236. ISSN: 0008-543X.
AU Nose H; Imazeki F; Ohto M; Omata M
AN 93306576 MEDLINE

L122 ANSWER 30 OF 115 HCAPLUS COPYRIGHT 2003 ACS
TI Expression of **human** p53 gene as glutathione S-transferase fusion proteins in Escherichia coli
SO Misaengmul Hakhoechi (1993), 31(4), 279-85
CODEN: MIHCAR; ISSN: 0440-2413
AU Oh, Sang Jin
AN 1994:185610 HCAPLUS
DN 120:185610

L122 ANSWER 31 OF 115 MEDLINE DUPLICATE 22
TI Alternative splicing of the p53 tumor suppressor gene in the Molt-4 T-lymphoblastic leukemia cell line.
SO CANCER LETTERS, (1993 Sep 30) 73 (2-3) 141-8.
Journal code: 7600053. ISSN: 0304-3835.
AU Chow V T; Quek H H; Tock E P
AN 94036762 MEDLINE

L122 ANSWER 32 OF 115 HCAPLUS COPYRIGHT 2003 ACS
TI Can **p53 peptides** serve as tumor-specific antigens?
SO Pept. 1992, Proc. Eur. Pept. Symp., 22nd (1993), Meeting Date 1992, 136-8. Editor(s): Schneider, Conrad H.; Eberle, Alex N. Publisher: ESCOM, Leiden, Neth.
CODEN: 60LUAN
AU Leder, Gerhard H.; Storkus, Walter J.; Zeh, Herbert J.; Lotze, Michael T.
AN 1995:66924 HCAPLUS
DN 122:78596

L122 ANSWER 33 OF 115 MEDLINE DUPLICATE 23
TI Characterization of cytotoxic T lymphocyte epitopes of a self-protein, p53, and a non-self-protein, influenza matrix: relationship between major histocompatibility complex peptide binding affinity and immune responsiveness to peptides.
SO JOURNAL OF IMMUNOTHERAPY, (1993 Aug) 14 (2) 121-6.
Journal code: 9102704. ISSN: 1053-8550.
AU Nijman H W; Houbiers J G; van der Burg S H; Vierboom M P; Kenemans P; Kast W M; Melief C J
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L122 ANSWER 34 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
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L122 ANSWER 95 OF 115 HCAPLUS COPYRIGHT 2003 ACS
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L122 ANSWER 115 OF 115 MEDLINE DUPLICATE 63
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=> d ab 5,8,16,32,35,36,43,56,57,73

L122 ANSWER 5 OF 115 MEDLINE DUPLICATE 3
AB The 90-kDa cellular protein encoded by the mouse mdm-2 oncogene binds to the p53 protein in vivo and inhibits its transactivation function (J. Momand, G. P. Zambetti, D. C. Olson, D. George, and A. J. Levine, Cell 69:1237-1245, 1992). cDNA clones encoding the **human** homolog of the mdm-2 protein (also called hdm-2) were isolated from a HeLa cell cDNA library. A series of monoclonal antibodies have been generated against **human** mdm-2 protein, and the epitopes recognized by these antibodies have been mapped. By construction of a series of deletion mutants, the region of the mdm-2 protein that is critical for complex formation with the **p53** protein has been mapped to the N-terminal **portion** of the **human** mdm-2 protein. Interestingly, a monoclonal antibody with an epitope located in this same region failed to immunoprecipitate the mdm-2-p53 complex and appeared to recognize only free mdm-2 protein. The domain of the p53 protein that is sufficient for interaction with **human** mdm-2 protein has been mapped to the N-terminal 52 amino acid residues of the p53 protein. This region contains the transactivation domain of p53, suggesting that mdm-2 may inhibit p53 function by disrupting its interaction with the general transcription machinery.

L122 ANSWER 8 OF 115 HCAPLUS COPYRIGHT 2003 ACS
AB An immune selection procedure was employed in order to isolate p53 binding sites from mouse genomic DNA. Two DNA clones capable of tight specific interaction with wild type p53 were subjected to further characterization. In both cases, the p53 binding regions displayed a high degree of sequence homol. with the consensus binding site defined for **human** genomic DNA. One of the clones was derived from the LTR of a retrovirus-like element (a member of the GLN family). The region encompassing the GLN LTR p53 binding site could confer p53 responsiveness upon a heterologous promoter. Furthermore, the expression of the endogenous, chromosomally integrated GLN elements was significantly induced upon activation of wild

type p53 in cells harboring a temp.-sensitive p53 mutant. Finally, it was demonstrated that p53-MDM2 complexes fail to bind tightly to such a p53 binding site. This may contribute to the inhibition by MDM2 of p53-mediated transcriptional activation.

L122 ANSWER 16 OF 115 MEDLINE DUPLICATE 12
AB The central role of the p53 tumor suppressor gene product in oncogenesis is gradually being clarified. Point mutations in the p53 tumor suppressor gene are common in most **human** cancers and are often associated with p53 protein overexpression. Overexpressed wild-type or mutant determinants of the p53 protein thus represent an attractive target for immunotherapy of cancer directed against a structure involved in malignant transformation. An important step towards this goal is identification of epitopes of p53 that can be recognized by **human** cytotoxic T lymphocytes. We identified **peptides** of (mutant) **p53** capable of binding to HLA-A2.1 in an in vitro assay. These HLA-A2.1-binding peptides were utilized for in vitro induction of primary cytotoxic T lymphocyte responses using a **human** processing-defective cell line (174CEM.T2) as antigen-presenting cell. These cells display "empty" HLA class I surface molecules, that can efficiently be loaded with a single peptide. We obtained CD8+ cytotoxic T lymphocyte clones capable of specifically lysing target cells loaded with wild-type or tumor-specific mutant **p53 peptides**. This strategy allows the in vitro initiation of **human** cytotoxic T lymphocyte responses against target molecules of choice.

L122 ANSWER 32 OF 115 HCAPLUS COPYRIGHT 2003 ACS
AB The authors screened the **human** p53 tumor suppressor sequence for HLA-A2 motifs and identified 10 nonameric peptide sequences with Leu or Met at position 2 and Ala, Ile, or Val at position 9. These **peptides** exhibited homol. solely to **p53** sequences of various species and not to other proteins listed in SwissProt. In addn., B-cell lines expressing HLA-A2 were loaded with these peptides and cell lysis was obsd. by a melanoma-derived infiltrating lymphocyte.

L122 ANSWER 35 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

L122 ANSWER 36 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

L122 ANSWER 43 OF 115 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.
AB Mutations in the p53 gene are most frequent in cancer. Many p53 mutants possess transforming activity in vitro. In cells transformed by such mutants, the mutant protein is oligomerized with endogenous cell p53. To determine the relevance of oligomerization for transformation, miniproteins containing C-terminal **portions** of **p53** were generated. These miniproteins, although carrying no point mutation, transformed at least as efficiently as full-length mutant p53. Transforming activity was coupled with the ability to oligomerize with wild-type p53, as well as with the ability to abrogate sequence-specific DNA binding by coexpressed wild-type p53. These findings suggest that p53-mediated transformation may operate through a dominant negative mechanism, involving the generation of DNA binding-incompetent oligomers.

L122 ANSWER 56 OF 115 MEDLINE DUPLICATE 36
AB Somatic mutation of the p53 gene is a very frequent event in the development of **human** neoplasia, and germ line mutations in p53 are responsible for an inherited cancer susceptibility syndrome. Many of the mutations in p53 found in **human** tumours are point mutations that result in the substitution of a single amino acid in the protein. These point mutant proteins are much more stable than the normal protein and the mutant product accumulates to a high level which permits important information about p53 expression to be obtained by immunochemical analysis. Using bacterial expression systems to produce **fragments** of **human p53** we have isolated and characterized new

monoclonal antibodies to p53. These antibodies are suitable for the measurement of p53 in ELISA, immunoblotting and immunoprecipitation analyses. They are especially useful in immunohistochemistry as they are able to react strongly with p53 in conventionally fixed and processed histological sections.

L122 ANSWER 57 OF 115 MEDLINE
AB Products of a number of mutant p53 genes bind with high affinity to members of the hsp70 family of chaperonin proteins, whereas wild type p53 lacks this type of association. Examination of the sequences of p53 genes from five different species enabled us to predict domains on p53 which may be involved in the association with hsp70 family members. A synthetic polypeptide (Pro-17-Gly) corresponding to the candidate hsp70 binding domain bound to in vitro translated hsp70 as determined by affinity chromatography and nondenaturing gel mobility shift assays. In addition, the Pro-17-Gly **peptide** competitively inhibited association between hsp70 and **p53**, an activity which was determined by immunoprecipitation with anti-p53 monoclonal antibody PAb240. The data indicate that p53 contains a hsp70 binding domain, which is located in a highly conserved region at the amino terminus of the protein, and may participate in the cellular function of wild-type p53 or in the transforming capacity of p53 mutants.

L122 ANSWER 73 OF 115 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 44
AB Synthetic peptide substrates for the cell division cycle regulated protein kinase, p34cdc2, have been developed and characterized. These peptides are based on the sequences of two known substrates of the enzyme, Simian Virus 40 Large T antigen and the **human** cellular recessive oncogene product, **p53**. The **peptide** sequences are H-A-D-A-Q-H-A-T-P-P-K-K-K-R-K-V-E-D-P-K-D-F-OH (T antigen) and H-K-R-A-L-P-N-N-T-S-S-S-P-Q-P-K-K-K-P-L-D-G-E-Y-NH2 (p53), and they have been employed in a rapid assay of phosphorylation in vitro. Both peptides show linear kinetics and an apparent K(m) of 74 and 120-mu-M, respectively, for the purified **human** enzyme. The T antigen peptide is specifically phosphorylated by p34cdc2 and not by seven other protein serine/threonine kinases, chosen because they represent major classes of such enzymes. The peptides have been used in whole cell lysates to detect protein kinase activity, and the cell cycle variation of this activity is comparable to that measured with specific immune and affinity complexes of p34cdc2. In addition, the peptide phosphorylation detected in mitotic cells is depleted by affinity adsorption of p34cdc2 using either antibodies to p34cdc2 or by immobilized p13, a p34cdc2-binding protein. Purification of peptide kinase activity from mitotic HeLa cells yields an enzyme indistinguishable from p34cdc2. These peptides should be useful in the investigation of p34cdc2 protein kinases and their regulation throughout the cell division cycle.

=> d ab 113

L122 ANSWER 113 OF 115 HCAPLUS COPYRIGHT 2003 ACS
AB Two synthetic peptides corresponding to residues 1-20 and 10-20, resp., of 1 type of a transformation-assocd., DNA- and antigen-binding cellular protein called p53 have been linked to a carrier protein and injected into rabbits to raise antibodies. The antibodies obtained were capable of reacting with the native protein, as judged by an ELISA, protein A-linked staining of immunoblots after SDS gel electrophoresis, and immunopptn. The immunoassay titers against the protein were lower for these antibodies than for antisera derived from immunization with purified p53. However, staining with the immunoblot method showed that the antipeptide antibodies against p53 were uniquely specific. The data suggest that at least 2 different types of p53 mols. occur. The cellular protein previously isolated from **human** cells transformed by Epstein-Barr virus and from murine tumors induced by methylcholanthrene appears to be larger than

the p53 reported in relation to simian virus 40- or adenovirus-transformed cells and to some other tumors. Some interrelationships have not been excluded, but it is clear that the 2 protein mols. do not behave identically. The reactions of the antipeptide antibodies with the intact protein have implications in regard to protein conformations. The strict specificities of such antibodies allow the generation of distinct sets of reagents useful for quantitation, purifn., and cloning.

=> d ab 1-4,13,16-19,23-26,28,30,33,35-40,42-44,46 l49

L49 ANSWER 1 OF 46 MEDLINE DUPLICATE 1
AB The serum response factor (SRF) is a 67-kDa phosphoprotein that, together with auxiliary factors, modulates transcription of immediate early genes containing serum response elements in their promoters. Here we show that the carboxyl-terminal domain of human SRF is phosphorylated in vivo and is recognized in vitro by the double-stranded DNA-activated serine/threonine-specific protein kinase, **DNA-PK**. SRF phosphorylation by **DNA-PK** was stimulated by its cognate binding site. Protein microsequence analysis of a 22-amino acid synthetic SRF peptide and phosphopeptide analysis of genetically altered glutathione S-transferase-SRF fusion proteins identified Ser-435 and Ser-446 of human SRF as sites phosphorylated by **DNA-PK** . Both serines are followed by glutamine. Changing Gln-436 and Gln-447 to other residues reduced or eliminated phosphorylation by **DNA-PK**, confirming that these glutamines are important determinants for kinase recognition. The carboxyl-terminal transcription activation domain was mapped within a 71-amino acid region that contains both **DNA-PK** phosphorylation sites. Amino acid substitutions that interfered with phosphorylation by **DNA-PK** at Ser-435/446 in GAL4-SRF fusion proteins were reduced in transactivation potency. From these data we suggest that **DNA-PK** phosphorylation may modulate SRF activity in vivo.

L49 ANSWER 2 OF 46 MEDLINE
AB Interaction of viral oncoproteins, such as SV40 large T, with cellular growth suppressor proteins Rb and p53 is presumed to inactive or modulate their growth suppression functions, thereby leading to transformation. An additional transformation-related activity of LT leads to hyperphosphorylation of p53. To search for kinases that might be responsible for this effect, p53-LT complexes were immunopurified from different SV40-transformed rat cell lines and assayed for associated kinase activities, in vitro. Protein kinase activity was readily observed in p53-LT immunecomplexes from wild-type transformed cells but was low or undetectable in p53 from mutant-transformed or normal cells. Optimal activity required the presence of Mn++. p53 was phosphorylated at all sites found in vivo. In contrast, LT was phosphorylated only at a subset of formerly identified sites and at additional sites not seen in vivo. The p53-LT-kinase complex was assayed for the presence of casein kinases, cdk like kinases, or DNA-activated kinase, using specific effectors, antibodies, or purified enzymes as tools. DNA-activated kinase or cdc2/cdk2 were not detectable, although the purified enzymes phosphorylated p53 in vitro. Casein kinase 2 represented the major activity, which on p53 phosphorylated not only the C-terminal Ser390 but also several sites in the N-terminal region. One additional activity, not identified so far, may represent an LT-induced or activated kinase. This kinase seems to enhance overall phosphorylation of p53 and, perhaps other substrates, and may thereby contribute to transformation.

L49 ANSWER 3 OF 46 MEDLINE DUPLICATE 2
AB Overexpression of wild-type p53 prevents cells from entering the S phase of the cell cycle. The amino-terminal transactivation region of p53 is phosphorylated by several protein kinases, including **DNA-PK**, a nuclear serine/threonine protein kinase that in vitro

requires DNA for activity. **DNA-PK** was recently shown to phosphorylate serines 15 and 37 of human p53 (Lees-Miller et al., 1992. Mol. Cell. Biol., 12, 5041-5049). To prevent phosphorylation at these sites, mutants were constructed that changed the codons for serine 15 or serine 37 to alanine codons. Expression of p53-Ala-37 in stably transformed T98G cells blocked progression of the cells into S phase as well as did the expression of wild-type p53. In contrast, p53-Ala-15 was partially defective in blocking cell cycle progression. Several cell clones transformed with the mutant p53-Ala-15 gene expressed normal levels of p53 mRNA but accumulated little or no detectable p53 protein. However, by using a transient expression system driven by a strong cytomegalovirus promoter, we showed that the inability of p53-Ala-15 to fully block cell cycle progression was not due to inadequate levels of expression or to a failure of the mutant protein to accumulate in the nucleus. These results suggest that phosphorylation of Ser-15 may affect p53 function.

L49 ANSWER 4 OF 46 MEDLINE DUPLICATE 3
AB The DNA-dependent protein kinase (**DNA-PK**) phosphorylates a number of transcription factors. Here, we show that the **DNA-PK** modifies c-Jun in vitro and that serine residue 249 (Ser-249) is required for phosphorylation to occur. This residue corresponds to one of three sites of c-Jun that are phosphorylated in vivo and which negatively regulate c-Jun DNA binding in vitro. However, we find that phosphorylation of c-Jun by the **DNA-PK** does not interfere with DNA binding, indicating that phosphorylation at other sites is required for this effect. Mutagenesis of the phosphorylated region of c-Jun reveals that the primary amino acid sequence recognised by the **DNA-PK** consists of the sequence Ser-Gln, and that adjacent acidic residues potentiate kinase activity. Furthermore, when this site is placed within the context of a second protein, it confers **DNA-PK** directed phosphorylation upon that protein. Our findings will facilitate identification of **DNA-PK** phosphorylation sites in other transcription factors.

L49 ANSWER 13 OF 46 MEDLINE

L49 ANSWER 16 OF 46 MEDLINE DUPLICATE 7

L49 ANSWER 17 OF 46 SCISEARCH COPYRIGHT 2003 THOMSON ISIDUPLICATE 8

L49 ANSWER 18 OF 46 MEDLINE DUPLICATE 9
AB The DNA-dependent protein kinase (**DNA-PK**) phosphorylates Sp1 and several other nuclear proteins. Here, we show that Sp1 and the **DNA-PK** must be colocalized on the same DNA molecule for efficient phosphorylation to occur. Interestingly, we find that the **DNA-PK** binds to and is activated by the ends of DNA molecules. Furthermore, we show that the DNA binding properties of the **DNA-PK** are identical to those of Ku, a well-characterized human autoimmune antigen. We demonstrate that the **DNA-PK** can be fractionated into two components, one of which is Ku and the other of which is a polypeptide of approximately 350 kd. DNA cross-linking and coimmunoprecipitation studies indicate that the catalytic 350 kd **DNA-PK** component is directed to DNA by protein-protein interactions with Ku. The implications of the unusual DNA binding mode and multicomponent nature of the **DNA-PK** are discussed.

L49 ANSWER 19 OF 46 MEDLINE DUPLICATE 10
AB Nuclear extracts prepared from human leukemic cells grown in the presence of cell differentiation-inducing agents showed a significant increase in the phosphorylation of a nuclear protein referred to as NP-72. The NP-72 phosphorylation was greatly increased by the addition of purified HeLa **DNA-PK** and was reduced by the inclusion of a **DNA-PK**-specific monoclonal antibody to the assay mixture.

Phosphoamino acid analysis showed serine (60%) and threonine (40%) residues to be the phosphate acceptors. Western blot analysis of control and TPA-treated nuclear extracts detected multiple immunoreactive protein bands. The two most prominent species migrated as 300- and 210-kD proteins on SDS-PAGE, possibly representing an intact and a processed form of **DNA-PK**. The 300-kD form of **DNA-PK** was only detected in TPA-treated nuclear extracts, raising the possibility that cell differentiation is associated with the down-regulation and/or the inhibition of a protease(s) capable of regulating **DNA-PK** turnover.

L49 ANSWER 23 OF 46 MEDLINE DUPLICATE 12
AB Alzheimer Disease (AD) is a distinct form of dementia characterized by the occurrence of neurofibrillary tangles, neurotic plaques and loss of certain neuronal populations. The tangles are associated with the presence of abnormal proteinaceous deposits. One such protein, referred to as tau, is found to be excessively phosphorylated in AD. We demonstrate that a double-stranded DNA-stimulated protein kinase (referred to as **DNA-PK**) effectively catalyzes the phosphorylation of recombinant human protein tau. Moreover, in the presence of stimulatory DNA, the hyperphosphorylation of tau is accompanied by a significant shift in its mobility on SDS polyacrylamide gels. These results suggest that **DNA-PK** may contribute to the pathogenesis of AD.

L49 ANSWER 24 OF 46 MEDLINE DUPLICATE 13
AB Human **DNA-PK** is a nuclear, serine/threonine protein kinase that, when activated by DNA, phosphorylates several DNA-binding substrates, including the tumor suppressor protein p53. To identify which p53 residues are phosphorylated, we examined **DNA-PK**'s ability to phosphorylate synthetic peptides corresponding to human p53 sequences. Serines 15 and 37 in the amino-terminal transactivation domain of human p53, and serines 7 and 18 of mouse p53, were phosphorylated by **DNA-PK** in the context of synthetic peptides. Other serines in these p53 peptides, and serines in other p53 peptides, including peptides containing the serine 315 p34cdc2 site and the serine 392 casein kinase II site, were not recognized by **DNA-PK** or were phosphorylated less efficiently. Phosphorylation of the conserved serine 15 in human p53 peptides depended on the presence of an adjacent glutamine, and phosphorylation was inhibited by the presence of a nearby lysine. Phosphorylation of recombinant wild-type mouse p53 was inhibited at high DNA concentrations, suggesting that **DNA-PK** may phosphorylate p53 only when both are bound to DNA at nearby sites. Our study suggests that **DNA-PK** may have a role in regulating cell growth and indicates how phosphorylation of serine 15 in DNA-bound p53 could alter p53 function.

L49 ANSWER 25 OF 46 MEDLINE DUPLICATE 14
AB Phosphorylation is an attractive mechanism for regulating the functions of p53. The p34cdc2 kinase, which is involved in regulation of the cell cycle, phosphorylates serine-315 of human p53 in vitro. Casein kinase II phosphorylates serine-389 of mouse p53 in vitro. The amino-terminal region of mouse p53 contains a cluster of potential serine phosphorylation sites. Those sites have been proposed to be sites for phosphorylation by a double-stranded DNA-dependent kinase (**DNA-PK**) from HeLa cells and can be dephosphorylated by protein phosphatase 2A. To identify in vivo phosphorylation sites in the amino-terminal region of mouse p53, we mutated potential phosphorylation sites and analyzed the mutant proteins by tryptic phosphopeptide mapping. We identified serine-7, -9, -18, and -37 as in vivo phosphorylation sites. We further showed that mouse p53 expressed in bacteria is phosphorylated by **DNA-PK** on amino-terminal serine residues in vitro.

L49 ANSWER 26 OF 46 MEDLINE DUPLICATE 15

AB Autophosphorylation of a **DNA-activated protein kinase** (**DNA-PK**) in Raji Burkitt's lymphoma cells generated a band that corresponded to a phosphoprotein of about 300 kDa on SDS/PAGE. This band corresponds to a 300-350-kDa **DNA-PK** found previously in HeLa cells. In addition to the 300-kDa phosphoprotein, the band of a highly phosphorylated 58-kDa protein was detected by SDS/PAGE of partially purified **DNA-PK** preparations after the phosphorylation reaction in the presence of double-stranded DNA. This phosphoprotein was specifically immunoprecipitated by phosphoprotein nor detectable activities of other kinases, phosphorylated recombinant c-Myc proteins in the presence of DNA. The c-Myc phosphorylation by **DNA-PK** was markedly stimulated by relaxed, double-stranded DNA, but neither by single-stranded DNA nor by RNA. Phosphopeptide mapping and phosphoamino acid analysis indicated that **DNA-PK** phosphorylates c-Myc in vitro at several serine residues.

L49 ANSWER 28 OF 46 MEDLINE DUPLICATE 16
AB The **DNA-activated protein kinase** (**DNA-PK**) is a nuclear serine/threonine protein kinase that phosphorylates DNA-binding proteins, including several transcription factors. **DNA-PK** is one of a very few enzymes known to be regulated through interaction with DNA that does not have DNA as a template or substrate. We suggest that **DNA-PK** may function in cell homeostasis, in part through the modulation of transcriptional activity.

L49 ANSWER 30 OF 46 LIFESCI COPYRIGHT 2003 CSA
AB The DNA-dependent protein kinase (**DNA-PK**) phosphorylates Sp1 and several other nuclear proteins. We show that Sp1 and the **DNA-PK** must be colocalized on the same DNA molecule for efficient phosphorylation to occur. We find that the **DNA-PK** binds to and is activated by the ends of DNA molecules. We show that the DNA binding properties of the **DNA-PK** are identical to those of Ku, a well-characterized human autoimmune antigen. We demonstrate that the **DNA-PK** can be fractionated into two components, one of which is Ku and the other of which is a polypeptide of approximately 350 kd. DNA cross-linking and coimmunoprecipitation studies indicate that the catalytic 350 kd **DNA-PK** component is directed to DNA by protein-protein interactions with Ku.

L49 ANSWER 33 OF 46 MEDLINE DUPLICATE 18
AB We have reported previously that chicken progesterone receptor (PR) is phosphorylated in vivo in response to progesterone administration. Three phosphorylation sites have been reported, two of which show increased phosphorylation in response to hormone and one which is phosphorylated only in response to hormone administration. We found previously that PR lacking the hormone-dependent phosphorylation is active in an in vitro transcription assay. Since the source of general transcription factors is a HeLa nuclear extract which contains many kinases, we have analyzed the receptor for phosphorylation during the in vitro transcription assay. We report here that the receptor is rapidly and efficiently phosphorylated on new sites, causing a change in receptor mobility on sodium dodecyl sulfate-gels. This phosphorylation is strictly dependent upon the presence of double stranded DNA. A **DNA-activated protein kinase** with similar properties has been isolated previously from HeLa cell nuclei. We find that phosphorylation of PR with this purified enzyme mimics the phosphorylation observed in the transcription assay. These data suggest that a previously undetected additional series of DNA-dependent phosphorylations may be required for activation of the PR.

L49 ANSWER 35 OF 46 MEDLINE DUPLICATE 19

AB Protein phosphorylation modulates the functions of simian virus 40 large T antigen (TAg) in productive and transforming infections. We recently described a **DNA-activated protein kinase** (**DNA-PK**) that efficiently phosphorylates TAg and several other nuclear, DNA-binding proteins in vitro (S.P. Lees-Miller, Y.-R. Chen, and C. W. Anderson, Mol. Cell. Biol. 10:6472-6481, 1990). In this report, we show by direct amino acid sequence analysis that **DNA-PK** phosphorylates TAg strongly at Ser-677, a residue known to be important for TAg interaction with origin site I and for transformation. We propose that **DNA-PK** may modulate the role of TAg in repressing early viral transcription and cell transformation, but a role for **DNA-PK** in regulating simian virus 40 DNA synthesis is not excluded. **DNA-PK** also phosphorylates Ser-665, and Ser-667, and one or more serines between amino acids 110 and 131. At least six serines, Ser-111, Ser-112, Ser-120, Ser-665, Ser-667, and Ser-677, are phosphorylated in TAg purified from baculovirus vector-infected insect cells.

L49 ANSWER 36 OF 46 MEDLINE DUPLICATE 20
AB **DNA-PK** is a moderately abundant serine/threonine protein kinase found in the nucleus of a wide range of eukaryotic cells. It is one of the few known cellular enzymes whose activity is regulated directly by DNA. Many DNA binding proteins, including a number of transcription factors, are substrates for **DNA-PK** in vitro. We suggest that this kinase may coordinate signal transduction pathways and nuclear events, including transcription, in response to changes in DNA or chromatin state.

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L49 ANSWER 38 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE 21

L49 ANSWER 39 OF 46 MEDLINE DUPLICATE 22
AB HeLa cells contain a serine/threonine protein kinase (**DNA-PK**) that is strongly activated in vitro by low concentrations of double-stranded DNA (dsDNA). Activation was specific for dsDNA; both natural DNAs and synthetic oligonucleotides functioned as kinase activators. The fact that **DNA-PK** activity was rapidly inhibited by incubation with dsDNA and ATP suggests that **DNA-PK** activity also may be regulated by autophosphorylation. During gel filtration, **DNA-PK** activity behaved as a 350-kDa protein, and highly purified **DNA-PK** contained a dsDNA-binding, 350-kDa polypeptide that was phosphorylated in a dsDNA-dependent manner. We conclude that this 350-kDa polypeptide is likely to be **DNA-PK**. Previously we showed that the dsDNA-activated kinase phosphorylates two threonines at the N terminus of hsp90 alpha (S. P. Lees-Miller and C. W. Anderson, J. Biol. Chem. 264:17275-17280, 1989). Here we show that **DNA-PK** also phosphorylates the simian virus 40 large tumor antigen, the mouse tumor-suppressor protein p53, the human Ku autoantigen, and two unidentified HeLa DNA-associated polypeptides of 52 and 110 kDa. Identification of these and other newly identified DNA-binding substrates suggest that the dsDNA-activated kinase may regulate transcription, DNA replication, or cell growth.

L49 ANSWER 40 OF 46 MEDLINE DUPLICATE 23
AB A **DNA-activated protein kinase** (**DNA-PK**) was purified from nuclei of HeLa cells. Activity was associated with a single high-molecular-mass (approximately-300,000 Da) polypeptide when analyzed by gel filtration, denaturing polyacrylamide gel electrophoresis, and Western immunoblotting using a monoclonal antibody that also inhibits enzyme activity. Nuclear

localization was indicated by subcellular fractionation and confirmed by immunofluorescence on whole cells. Double-stranded DNA stimulated phosphorylation of the 300-kDa polypeptide in purified preparations as well as phosphorylation of the exogenous substrates alpha-casein, simian virus 40 large T antigen, and the human heat shock protein hsp90. Autophosphorylation led to inactivation of the enzyme. The phosphorylation of casein was stimulated over 30-fold by DNA and was specific for serine and threonine residues. Bovine serum albumin and histone H1 were poor substrates for **DNA-PK**, and no phosphorylation of immunoglobulin G or histones other than H1 was observed. Supercoiled or heat-denatured DNA and synthetic double-stranded RNA or RNA-DNA copolymers did not stimulate casein phosphorylation by **DNA-PK**. Interaction of the enzyme with DNA in the absence of exogenous substrates was demonstrated by thermal inactivation and gel mobility shifts. These characteristics identify **DNA-PK** as distinct from other protein kinases described in the literature and suggest that activation by DNA is an important feature of the enzyme's in vivo function.

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L49 ANSWER 43 OF 46 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

L49 ANSWER 44 OF 46 MEDLINE DUPLICATE 24
AB The 90-kDa heat-shock protein, hsp90, is an abundant cytoplasmic protein that can be phosphorylated in vitro by a double-stranded (ds) **DNA-activated protein kinase** found in cells from several species. Here we show that the dsDNA-activated protein kinase from human HeLa cells phosphorylates 2 threonine residues in the sequence PEETQTQDQPME at the amino terminus of human hsp90 alpha. Hsp90 beta, which is 97% identical to hsp90 alpha but lacks both amino-terminal threonines, is not phosphorylated by the dsDNA-activated protein kinase. Mouse hsp86 and rabbit hsp90 alpha are homologous to human hsp90 alpha; both heterologous proteins are phosphorylated at the same amino-terminal threonines by the human dsDNA-activated protein kinase.

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AB Short communication.

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AN 1991(13):02854 NTIS Order Number: DE90016556/XAB
TI SV40 large tumor antigen and the tumor suppressor p53 are phosphorylated by a **DNA-activated protein kinase** from human cells.
AU Lees-Miller, S. P.; Chen, Y. R.; Anderson, C. W.
CS Brookhaven National Lab., Upton, NY.
Sponsor: Department of Energy, Washington, DC. (004545000 0936000)
NR DE90016556/XAB; BNL-43799, BIO-4569; CONF-9007160-1
4p; 1990
NC Contract(s): AC02-76CH00016
DT Report
CY United States
LA English
NTE International conference on methods in protein sequence analysis (8th), Kiruna (Sweden), 1-7 Jul 1990. Sponsored by Department of Energy, Washington, DC.
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